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TI Store depletion triggers the calcium release-activated calcium current (ICRAC) in macrovascular endothelial cells: a comparison with Jurkat and embryonic kidney cell lines.

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AB In endothelial cells, different types of Ca²⁺ conductances have been described, but none of them has been clearly identified as ICRAC, the Ca²⁺

release-activated Ca²⁺ current originally described in mast and lymphoma cells. Here we show that in bovine pulmonary artery endothelial cells (CPAE) depletion of intracellular Ca²⁺ stores by inositol 1,4,5-trisphosphate (InsP₃), Ca²⁺ ionophores and Ca²⁺ pump inhibitors activates a Ca²⁺-selective conductance in the presence of the Ca²⁺ chelator 1,2-bis(2-aminophenoxy)ethane-N,N,N', N'-tetraacetic acid (BAPTA). The current shows inward rectification, a highly positive reversal potential and is blocked by micromolar concentrations of La³⁺. The conditions used in studies of endothelial cells were also employed in those of HEK-293, an **embryonic** kidney cell line commonly used to express putative store-operated channels, and Jurkat cells, the **reference cell** model. Similar to CPAE, HEK cells also have an ICRAC-like current. At 0 mV

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L1 728 S REFERENCE (W) CELL?
L2 11 S L1 (P) EMBRYO?
L3 0 S L2 (P) (ADVANTAG? OR USEFUL? OR PREFERR?)
L4 5 DUPLICATE REMOVE L2 (6 DUPLICATES REMOVED)